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GASTROCNEMIUS MEDIALIS MUSCLE ARCHITECTURE AND PLANTARFLEXOR TORQUE PRODUCTION IN POSTSTROKE SURVIVORS

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SUMMARY

The purpose of this study was to compare the architecture of the medial gastrocnemius muscle and the plantarflexor torque between stroke survivors with ankle spasticity and healthy subjects. The study included 15 stroke survivors and a control group of 15 healthy subjects. An isokinetic dynamometer was used for the plantarflexor torque evaluation, while images of the medial gastrocnemius muscle were obtained using ultrasonography. Images were collected at rest and during a maximum voluntary isometric contraction at 0° (neutral position) of plantar flexion. The unaffected limb displayed a similar muscular structure to control but with impairments in the ability to produce force. The affected limb presented smaller fascicle length at rest and smaller pennation angle and reduced peak torque during the maximal isometric voluntary contraction compared to control and unaffected limbs.

INTRODUCTION

Spasticity and muscle weakness are commonly observed in stroke survivors affecting life quality [1]. Muscle architecture is related to contractile properties and, consequently, to muscle force production [2]; however, only few studies have looked at spastic muscles from stroke survivors using ultrasound images [3]. As muscle force production depends on muscle architecture [4], it will have implications in the mechanical properties of spastic muscles. Thus, the aim of this study was to compare the gastrocnemius medialis muscle (GM) architecture at rest and during maximal voluntary contraction (MVC) of stroke survivors with ankle spasticity with that of healthy subjects.

METHODS

Fifteen stroke survivors with ankle spasticity (55.9 ± 9.3 years old, 77.1 ± 14.5 kg of body mass, and 169 ± 0.06 cm of height) and fifteen healthy subjects (58.7 ± 6.6 years old, 74.6 ± 10.8 kg of body mass, and 168 ± 0.10 cm of height) signed an informed consent form to participate in the study, which was approved by the University Ethics Committee in Human Research. Ashworth Scale [5] was used to quantify ankle spasticity (1.5 ± 0.6). Body mass, standing height and lower limb length were measured in all subjects. Subjects were sat on the isokinetic dynamometer chair (Biodex Medical System, Shirley, NY, USA) where the knee joint remained fully extended and the ankle joint was kept neutral (0°). Ultrasound images were gathered from the GM using an ultrasound scanner (SSD 4000, 51 Hz, ALOKA Inc., Tokyo, Japan) at 50% of the muscle belly length at rest and during MVC at 0° of ankle plantar flexion. Subjects were instructed to relax and to perform a maximal isometric plantar flexion for architectural measurements. A synchronization unit (HORITA Video Stop Watch VS-50; HORITA Co. Inc., California, USA) was used to synchronize the isokinetic dynamometer data with ultrasound scanner data. The affected limb was evaluated followed by the unaffected limb for stroke survivors. The dominant limb of healthy subjects was used as control. GM fascicle length was determined assuming a linear distance between the insertions of the muscle fascicle to the superficial and to the deep aponeuroses and pennation angle was defined as the angle between the fascicle and the deep aponeurosis. Fascicle lengths were normalized by lower limb length. All images were analyzed in ImageJ (National Institute of Health, USA) software. Peak torque was defined as the maximum torque computed during the 5-sec effort and was normalized by body mass.

RESULTS AND DISCUSSION

There were no significant differences between groups (stroke survivors vs. healthy) for age (p=0.28), body mass (p=0.63) and standing height (p=0.60). There were no differences (p=0.43) in fascicle length for the affected limb (0.09±0.01cm/cm) compared to the unaffected limb (0.11±0.02 cm/cm) at rest. There were no differences (p=0.61) comparing the unaffected limb to control (0.12±0.02cm/cm). However, shorter fascicle lengths were observed for the affected limb compared to the control (p=0.02). At MVC, the unaffected limb (0.07±0.02cm/cm) presented shorter fascicle length compared to the affected limb (0,08±0,02cm/cm) (p=0.01). Fascicle length of the affected and unaffected limbs did not differ from the control (0.07±0.01cm/cm) (p=0.23). During rest, pennation angle did not differ (p=1.00) comparing affected (17.5±4.0) to unaffected (18.2±2.6) limbs. Both the affected limb and the unaffected limb did not differ (p=0.89) from the control (18.9±3.9). During MVC, the affected limb showed smaller pennation angle (21.5±5.7) compared to the unaffected limb (30.6±6.6) and to the control (31.0±5.9) (p<0.01). There were no differences (p=1.00) between the unaffected limb and the control. Affected limb (0.77±0.4Nm/kg) presented reduced peak torque (p<0.01) than the unaffected (1.20±0.3Nm/kg) and the control limbs (1.78±0.4Nm/kg). Greater peak torque was found for the control compared to the unaffected limb (p<0.01) (Figure 1).

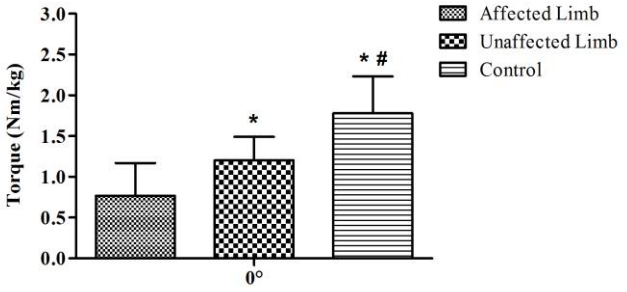

Figure 1: Net ankle torque normalized by body mass obtained at 0° of plantar flexion for the affected and unaffected limbs of stroke survivors and for the control subjects; ***:** differences to affected limb; **#:** differences to unaffected limb.

The similarity in fascicle length between the affected and the unaffected limbs observed at rest is in agreement to findings from Malaya et al [6]. However, the shorter fascicle length for the unaffected limb during MVC may reflect the neurological problems that do not allow the patients to fully activate and shorten muscle fascicles in spastic muscles [7]. Shorter fascicle length was observed comparing the affected limb to the control. Similar to muscle immobilization at shorter lengths, spasticity may be linked to reduced sarcomeres in series due to lower mechanical demand [8]. During MVC, muscle fascicle length did not differ between stroke survivors and control. Despite the longer fascicle length in control, this result may be explained by the greater ability to shorten the fascicle in this group. For pennation angle, stroke seems to impair subjects to fully activate the plantar flexor muscle group [7], which may explain the reduced pennation angle and peak torque during MVC in the affected limb compared to unaffected. However, differences were only observed at MVC for affected and control, suggesting a reduced capability for increasing pennation angle due to reduced muscle length in stroke survivors. There were no differences in muscle architecture comparing the unaffected limb to the control for rest and MVC. These results are in disagreement to others that showed differences in muscle architecture of the unaffected limb to control [9]. Furthermore, the control produced more torque compared to the affected and unaffected limbs, which may be related to reduced number of motor neurons [10] in affected limb and for the unaffected limb also presenting affected cortical areas that remain ipsilateral reducing neural drive and limiting force production [11].

CONCLUSIONS

The unaffected limb appears to have a muscular structure similar to control, but it showed impairments in the ability to produce force. The affected limb presented smaller fascicle length at rest and muscle function also differed from control and from the unaffected limb due to reduced peak torque and smaller pennation angle during the maximal isometric voluntary contraction.

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